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"Osteochondrosis in Swine"

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Summary

Osteochondrosis is a well defined clinical syndrome causing acute lamenesses of sudden onset in European swine, especially the European Landrace. It is characterized as:

(1) following familial lines.
(2) occurring primarily at 6-12 months of age.
(3) relating to certain conformations.
(4) unrelated to stress or nutritional levels of calcium, phosphorous, Vitamin A, Vitamin D, and protein.
(5) occuring in fast growing swine. Those with the genetic ability to grow and/or high-energy rations.

With the emphasis presently being placed on soundness in the American swine industry, producers are showing an increasing interest in finding out why lamenesses occur instead of just marketing the animals involved. Several animals seen at the Iowa State College of Veterinary Medicine in the past few months have shown typical clinical signs and post mortem lesions as described in European literature. Osteochondrosis has probably been a problem in the American swine industry for years, but is only now being recognized and should be considered in the differential diagnosis of acute lameness conditions in swine, especially those of breeding age.

Osteochrondrosis is a generalized condition occurring in many joints of the body including those of the limbs and vertebral column, and even involving costochondral junctions, olecranon, and ischial apophyses. The term is presently being applied to a number of poorly defined "leg weakness" syndromes in swine. These syndromes are characterized by acute lamenesses usually in 6–12 month old animals and can be very costly to the swine producer. It is most common in the forelegs. The major difference between the syndrome in Europe and that seen in the United States is the age of onset. In Europe the condition is commonly seen in swine 4-7 months of age while in the United States it is primarily seen in 6-10 month old pigs. The reason for this is not completely clear, but Dr. Jerry Kunesh (Iowa State Ambulatory Clinic) points out that many

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producers have no problem when marketing at 200-230 pounds, but when hogs are held to 240-260 pounds they have lameness problems which would tend to indicate that the incidence may be higher than previously suspected.

In Swedish studies the "leg weakness" syndrome was investigated in over 200 Yorkshire and Landrace boars with the following findings given in order of decreasing incidence:

(1) Osteochondrosis—referring to a disturbance of endochondral ossification and osteogenesis often leading to osteochondritis dissecans (which refers to aseptic degeneration of articular cartilage) and to epiphysiolysis.
(2) Osteoarthrosis—either primary or secondary degenerative disease which may follow osteochondrosis.
(3) Arthritis—an acute or chronic infectious inflammation.

In the Swedish studies osteochondrosis occurred in swine less than 18 months of age and was most prevalent in the 6-12 month range. Swine less than five months had no cartilage defects, but showed yellow areas of beginning degeneration. Swine 6-12 months of age showed actual defects and severe lameness. Twelve-eighteen month old swine were apparently recovered with scar tissue filling the lesions. This high incidence of acute lameness in swine 6-12 months of age occurs when weight gains are rapid, and may relate to the fact that swine, unlike other mammals, have a long period between sexual maturity at 5-6 months and skeletal maturity at 18-20 months. The lesions found, in order of incidence, are as follows:

(1) Thickened, irregular epiphyses with no ingrowth of vessels or calcification.
(2) Progressive degeneration of the articular cartilage leading to cracks and crevices with the development of cartilage flaps and free joint bodies.
(3) Premature closure of growth plates leading to deformation of bone.
(4) Epiphysiolysis, which refers to separation of the epiphysis and metaphysis.

The finding of healed lesions in older animals is supported by Dr. David Graham (Iowa State College of Veterinary Medicine, Department of Pathology) who acknowledges posting a number of older swine in which he found healed lesions filled with granulation tissue. This may explain why the severe condition suddenly crops up in certain breeds or families where it has not been a major problem previously.

Osteoarthrosis, unlike osteochondrosis, was generally found in swine greater than 18 months and was usually found in the small joints of the hocks. It was typically bilateral and symmetrical. The articular cartilage varied from yellow and thickened, with decreased synoviae to pits, erosions, hyperemic granulation, and ankylosis.

In the Swedish study previously mentioned, macro- and microscopic studies were made with the following observations occurring often enough to be considered significant.

The shoulder often showed cartilage damage with the caudal portion of the head of the humerus showing an irregular, oval defect, with a rounded rim and reddish floor as is seen in osteochondritis dissecans.

The elbow joint showed pathological changes on the cartilage of the medial condyle which appears flattened in the caudal and medial portions with the central ridge of the lateral condyle varying in shape and size. The anconeal process is often pushed laterally and has lesions on its medial surface. The incidence of these lesions was related to the width of the semilunar notch and the direction of the guiding ridges on the radius and ulna.

In the stifle joint, the medial femoral condyle was often flattened and less developed than the lateral. The central part of the medial condyle cartilage was pathological, wrinkled, loose, and occasionally detached and replaced by fibrocartilage. These lesions were usually bilateral and symmetrical.

The medial phalanx was often considerably shorter than the lateral both fore and back with the medial hoof being smaller.

Many of these European pigs with these foreleg lesions were also "knock-kneed" and, as previously mentioned, and small inside toes. This condition is being seen more and more in the American swine industry and could be viewed at most large swine operations. Such a conformational defect would result in undue stress on the various

joint surfaces of the forelegs.

These observations were supported by conformational studies done at the Veterinary College of Norway in Oslo. In these studies, swine with narrow lumbar and pelvic regions and heavy muscling of the inner stifle were found to have a higher incidence of lesions. This could be related to the angle of the femur in relation to the pelvis and tibia. Since conformation in swine is considered to have up to 50% heritability, this would tend to support a genetic basis for the disease.

The Swedish study touched upon the genetic theory in that they investigated the incidence of osteochondrosis in fast-growing strains of swine. When the Swedish crossed fast-growing, osteochondrosis-prone lines of domestic swine with wild swine, the bone growth rate and the incidence of osteochondrosis decreased. There may thus be a correlation with levels of somatotropin. In 1971, Lundgren was able to correlate the incidence of osteochondritis dissecans in dogs with hormone imbalances leaning toward excess somatotropin. It has also been found that osteochondritis dissecans in man with slipped capital femoral epiphysis is common in individuals with endocrine dysfunction. Since STH affects primarily chondrogenesis and secondarily osteogenesis, it would seem a plausible explanation.

A variety of studies have also been done in an attempt to relate the incidence of osteochondrosis to nutrient levels. These studies were initiated because growth plate changes were in some respects similar to those of rickets seen in young bulls on diets deficient in calcium and/or Vitamin D. The Swedish tested varying diets of calcium, phosphorous, Vitamin D, Vitamin A, and protein. Neither deficiencies or excesses of these dietary components influenced the incidence of osteochondrosis. It was found, however, that growth rate affected the incidence of osteochondrosis. When litters were divided with some being fed *ad libitum* and others being on controlled intake, the slower gaining pigs had significantly less osteochondrosis. In another study reported upon by T. D. Tanksley, Jr., at the International Pig Veterinary Society, 1976, it was found that levels of calcium and phosphorous for maximum gain and feed efficiency do not correspond with levels for maximum bone development by bone breaking strength. While NRC requirements of .75% calcium and .50% phosphorous were sufficient for growth, levels of 1.2% calcium and .74-1.0% phosphorous were needed for maximum bone development.

Other etiologic theories which have been offered include the stress-mechanical influence theory. While stress in general and excess mechanical stress in joints in particular could theoretically trigger the condition, this theory offers no explanation for the high incidence of osteochondrosis in costochondral junctions and apophyses which are non weight bearing. Thus, osteochondrosis may well be the result of a generalized condition having effects on basic processes and events of subchondral ossification and joint cartilage physiology and multiple etiological factors, including nutrition, growth rate, conformation, and genetics may all contribute to its pathogenesis.

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